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Viral hepatitis (Invited Presentation)

13.001

Epidemiology of Chronic Viral Hepatitis in Latin America

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Chronic viral hepatitis caused by Hepatitis B or C viruses are major health problems in this beginning of the 21st century. Estimated prevalence in the world population in different regions range between less than 1% to more than 3% for HCV and between less than 2% to more than 8% for HBV, affecting more than 400 million people in the world. In Latin America, prevalence estimates are flawed. For HCV it varies from less than 1% to 2%, and for HBV from less than 1% to more than 8%. Numbers can be as high as 15% in the Amazon region.

In Latin America, some surveys report HBV prevalence as high as 21.4% in Dominican Republic and 7.9% in Brazil, followed by 3.2% in Venezuela and 2.1% in Argentina. Low prevalence was found in Mexico (1.4%) and Chile (0.6%). For HCV, rough estimates project more than 10 million infected people. Many surveys were conducted by blood banks, but results are biased by sampling problems.

In Brazil, HCV prevalence studies estimates had found a wide range, varying from 0.4% to 5.9%. A population based study in 2007 found a HCV antibodies prevalence of 0.28% to 2.61% and a HCV-RNA from 0.02% to 0.9% in different regions of the country. From 1994 to 2005, the Ministry of Health database has registered 52,440 HCV cases. Recently, a national survey was conducted by the Ministry of Health, but results are not published yet.

In São Paulo state, there were 30,299 HCV cases registered from 2002 to 2008 and 14,810 HBV cases in the same period. In the city of São Paulo it is estimated a mean prevalence of 1.42% (95% confidence interval 0.7 — 2.12%). Diagnosis can be done with blood tests, but availability is a concern in poor countries.

Treatment is expensive and fairly effective, implying in high morbidity, mortality and costs. Chronic hepatitis is a great challenge for the health systems in Latin America.

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13.002

Update on Hepatits B Therapy

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Tha main goal for the treatment of chronic hepatitis B (CHB) is to prevent advanced hepatic disease: cirrosis,hepatic failure and hepatocellular carcinoma (HCC). The first aim of treatment is to achieve sustained suppresion of HBV replication as well as the remission of liver disease.The sustained supression of virological replication from genotype. Since 1992,eigth therapeutic agents have been approved worlwide (INF alfa, lamivudine, adefovir, entecavir, PegINF alfa-2ª thymosin alfa1, ,telvibudine and tenofovir) but only some of them are used in different countries according to national regulation.. When and how to treat an CHB depends on the HBV DNA levels, ALT and status of HBeAg. For HBeAg(+) patients,the endpoint of treatment is HBeAg seroconvertion. Therapy is considered in GHB with HBV DNA leves of 20.000 IU/ml or higer (HBeAg positive patient) or 2.000 IU/ml (HBeAg negative), although lower HBV DNA levels migth be selected when evidences of progresive disease are identified. ALT normalization and HBV DNA suppression are the measures of reponse to therapy. Oral nucleoside analogs (NA) is a significant contribution for treatment in the last years,but a major concern with this agents is the selection of antiviral resistant mutations. This may be identified prior to virological breakthrough or at the same time. Peginterferon alfa-2ª, entecavir and tenofovir are currently included in the first-line treatment choice on the basis of their potency as well as the low rate of antiviral drug resistance. The strategy of drugs combination in CHB treatment for achieving a sustained virological response and some end points has beeen explored and the level of HBV DNA suppression. This combination theraphy is encouraging in some clinical trials.

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13.003

Hepatitis C Treatment Today and the Future

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Therapy of chronic HCV infection is based on the use of the combination of pegylated interferon and ribavirin. Sustained virological response (SVR), a negative HCV RNA 24 weeks following discontinuation of therapy, is the most important surrogate parameter to achieve. Actually, SVR is obtained in about 50% of patients with genotypes 1/4 and in 80% of the patients with genotypes 2/3.

Patients infected with genotypes 1 or 4 must be treated for 48 weeks. But, if the patient achieves a rapid virological response (RVR), defined as a negative HCV RNA at week 4, we can consider a shortening of treatment. In patients with a slow response to treatment (HCV RNA only negative between weeks 12 and 24) the length of therapy must be extended to 72 weeks. For patients infected with genotypes 2 or 3 treatment should be planned for 24 weeks.

New drugs are needed for non-responders and for those who are not good candidates to treatment.

Several new oral agents, more potent, less toxic and allowing for shorter duration of treatment are being developed. These new drugs are designed to inhibit several viral enzymes. Results of recent clinical trials using inhibitors of NS3/4A protease or inhibitors of NS5B polymerase in combination with peginterferon/ribavirin are promising. These studies demonstrated that adding telaprevir or boceprevir (the protease inhibitors in the most advanced phases of evaluation) to peginterferon/ribavirin improved the rates of SVR

in treatment-naïve and treatment-experienced patients. From these and other trials it was possible to conclude that the use of these new agents in monotherapy, owing to its relative low genetic barrier, was associated with a rapid development of resistance to the drugs and that the use of ribavirin was always necessary. These new agents will be available for general clinical use in the next years but they must be used as a complement of current therapy.

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13.004

Management of HIV and Hepatitis C Co-infection

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The rate of coinfection of HIV with Hepatitis C is high in countries where the mode of transmission is predominantly intravenous drug abuse. The success of highly active antiretroviral therapy (HAART) in decreasing HIV related morbidity and mortality has shifted the focus of care for people living with HIV. More attention is being paid to the management and prevention of chronic ailments such as cardiovascular, liver and renal disease. Thus, it is important for the clinician treating HIV infected patients to recognize the clinical presentations, spectrum of disease, efficacy of treatment and principles of management for coinfected patients. Patients coinfected with the HIV and Hepatitis C viruses have an increased risk of liver related morbidity and a more rapid progression to end-stage liver disease. The treatment of these patients is complex owing to the significant side effects and limited efficacy of Peg Interferon and Ribavirin. This lecture will review the epidemiology, natural history, diagnosis, management and newer treatment modalities in HIV/HCV coinfection.

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Infectious diseases following catastrophes (Invited Presentation)

14.001

Infectious diseases and infection control after natural disasters

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Infections are frequent complications after natural catastrophes. Previous reports suggest a high prevalence of colonisation and infection with multi-resistant Gram-negative pathogens in victims of natural disasters.

Literature regarding infections and infection control measures after natural disasters was reviewed from 1986 through the end of 2009, with special emphasis on the 2004 tsunami. Local microbiology of patients followed in our institution was also reviewed.

Patients admitted after natural disasters often have polymicrobial infections with atypical bacteria and fungi. Moreover, they are usually colonised or infected with multi-drug resistant organisms. These pathogens are acquired either nosocomially or environmentally. Several studies have suggested that Gram-negative bacteria are more prevalent than Gram-positive bacteria. A high incidence of colonisation and infection with extended spectrum β-lactamase-producing bacteria, multi-resistant non-fermenting Gram-negative rods and difficult to treat fungal infections are found in these patients and may pose challenges in routine hospital care.

According to published date and our own experience, we recommend pre-emptive contact isolation for victims of natural disasters during hospitalisation until results of microbiological cultures become available. If respiratory symptoms are present, droplet isolation must be included. These measures should also be applied during the air transportation of these patients. Considering the different multi-resistant colonisers, cohorting patients must be avoided whenever possible. In cases of life-threatening infections, empiric antibiotic therapy must cover multi-resistant non-fermenting Gram-negative rods. Clinicians must be aware of unusual microbiological findings in these patients.

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14.002

Infectious diseases and war conflicts in the Middle East

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Infectious diseases and war have been witnessed for as long as human life. Historically, infectious diseases have been responsible for the majority of deaths during war; however, numerous medical and military advances have reversed this trend, resulting in more deaths from battle than infectious diseases in the 20th century. Wounds incurred in war are grossly contaminated with bacteria and most will become infected unless appropriate treatment is initiated quickly.Common infections include respiratory as well as gastrointestinal infections. Endemic diseases are also reported during the war and they include Brucella, Q-fever, Malaria, Sandfly fever and Leishmaniasis. Non-battle injuries such as mental and combat stress are common; while battle associated infections such as trauma- related complications are extensively reported.

Multidrug resistances (MDR) Gram negative bacilli have been reported in war wound infections, particularly Acinetobacter spp, Enterobacter spp.and Pseudomonas spp. and therefore empirical treatment for infected war wounds should be given to cover MDR. Other war related infections such as malaria, MDR tuberculosis, chronic Q fever and brucellosis may become apparent after returning home and therefore they should be considered due to their lengthy reactivation periods. In addition to this, vaccines have proven to be an important breakthrough to help prevent the spread of several infectious diseases.

War wounds are predisposed to infection due to environmental conditions on the battlefield, devitalized tissue, and foreign bodies in the wound as well as delays in evacuating causalities. Knowledge of likely pathogens for particular infections and sites, as well as optimal antibiotics to eradicate those pathogens will aid battlefield